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L8   ANSWER 1 OF 11  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Adult stem cell ***recruitment*** in an animal using a protease or an
     activator of a protease, and a method of treating myeloproliferative
     disorder
AB   The invention relates to the use of proteases to ***recruit***  stem
     cells from the niches they normally occupy, for example, from the bone
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marrow. For example, in MMP9-/- mice, release of Steel-encoded Kit-ligand (sKitL) and hematopoietic stem cell motility are impaired, resulting in failure of hematopoietic recovery and increased mortality. As described herein, MMP9 (matrix metalloproteinase-9/gelatinase B) enables bone marrow repopulating cells to translocate to a permissive vascular niche favoring differentiation and reconstitution of the stem cell and progenitor cell pool. Thus, the invention is directed to a method for ***recruitment*** of adult stem cells in an animal comprising administering to the animal a protease or an activator of a protease, wherein the ***recruitment*** translocates an endogenous population of quiescent non-cycling stem cells to a permissive vascular zone in the animal so that the stem cells can proliferate, self-renew, differentiate or mobilize to a target site. The activator can be interleukin-1, thrombopoietin, ***G***, -, ***CSF***, GMCSF, SDF-1, or ***fibroblast*** growth factor-4. The protease can be a matrix metalloproteinase, a collagenase, a gelatinase, a stromelysin, a matrilysin, a metalloelastase, or a membrane-type matrix metalloproteinase. The method may also be used to induce proliferation of adult stem cells in an animal. A therapeutic method for treating a myeloproliferative disorder in a mammal using the same is claimed.

SO U.S. Pat. Appl. Publ., 77 pp.
CODEN: USXXCO

L8 ANSWER 2 OF 11 CAPLUS COPYRIGHT 2008 ACS on STN

TI Device compositions for the ***recruitment*** of cells to blood contacting surfaces in vivo

AB Methods and compns. for ***recruiting*** cells circulating in the blood stream of a subject to a blood contacting surface, and in particular, devices and methods for ***recruiting*** endothelial cells to a blood contacting surface of a prosthesis as well as engineering a self-endothelializing graft in vivo by ***recruitment*** of circulating endothelial progenitor cells (EPCs) to form a neo-endothelium on a prosthetic structure are described. Examples are given for ***recruitment*** of endothelial progenitor cells to a blood contacting surface by ligand interaction and by magnetic interaction in vivo.

SO U.S. Pat. Appl. Publ., 30 pp.
CODEN: USXXCO

L8 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2008 ACS on STN

TI IL-17

AB A review. IL-17 is a potent proinflammatory cytokine produced mainly by activated memory CD4+ T cells. The family of IL-17, a new family of cytokines, is composed of six functionally related members, ie, IL-17 and IL-17B-F in humans and mice. IL-17 exerts its biol. activity as a homodimer. In contrast to the selected expression pattern of this gene, the IL-17 receptor is ubiquitously distributed among diverse tissues and cells. IL-17 induces the secretion of IL-6, IL-8, PGE2, MCP-1 and ***G***, -, ***CSF***, by ***fibroblasts***, keratinocytes, epithelial and endothelial cells, and is also able to induce ICAM-1 expression, T cell proliferation, and growth and differentiation of CD34+ human progenitors into neutrophils. The involvement of IL-17 in the rejection of allogeneic grafts has been demonstrated. The potent inflammatory actions that have been identified for IL-17 and the emerging assocns. with major human diseases, such as rheumatoid arthritis and allergic asthma, suggest that the family of IL-17 may have significant roles in the pathophysiol. of inflammatory processes. IL-17 induces prodn. of metalloproteinases and nitric oxide, responsible for the aggravation of arthritis and joint destruction. IL-17 can ***recruit*** and activate neutrophils in the airways, mediated by IL-8 and MIP-2. In addn., IL-17 stimulates human bronchial epithelial cells to release the neutrophil-activating factor IL-6.

SO Biotherapy (Tokyo, Japan) (***2003***), 17(1), 85-97
CODEN: BITPE9; ISSN: 0914-2223

L8 ANSWER 4 OF 11 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI Multiple Mechanisms of SOCS3-Mediated Inhibition of ***G*** - ***CSF*** -Signaling Are Disrupted by C-Terminal Truncation of ***G*** - ***CSF*** Receptors Found in SCN/AML.
AB ***G*** - ***CSF*** receptor (G-CSFR) signaling regulates survival, proliferation and differentiation of myeloid progenitor cells into neutrophils. Different domains and four conserved tyrosine residues in the receptor C-terminus (Y704, Y729, Y744, Y764) have been linked to signal transduction pathways that mediate these effects. Suppressor of cytokine signaling (SOCS) proteins utilize their SH2 domains to inhibit signaling from both critical tyrosine residues in receptor chains and intermediate signaling molecules. Members of the SOCS protein family implicated in negative feedback during hematopoietic growth factor signaling are CIS, SOCS1, SOCS2 and SOCS3. Whether these proteins downregulate ***G*** - ***CSF*** signaling and which domains in the G-CSFR are involved is still unclear. Also, it is unknown whether and how mutations truncating the G-CSFR found in severe congenital neutropenia (SCN) patients with a predisposition to AML affect SOCS-mediated downregulation of ***G*** - ***CSF*** signaling. We first investigated which SOCS proteins were capable of inhibiting ***G*** - ***CSF*** -induced STAT5 activity in HEK293 cells. We found that SOCS1 and SOCS3, but not CIS and SOCS2, inhibited G-CSFR activity. Experiments with tyrosine "null" (m0) and single tyrosine add-back mutants revealed that while SOCS1-mediated inhibition of ***G*** - ***CSF*** signaling was entirely independent of C-terminal G-CSFR tyrosines, Y729 is a major ***recruitment*** site for SOCS3-mediated inhibition of ***G*** - ***CSF*** responses. The fact that the Y729 motif is universally lost in SCN-derived ***G*** - ***CSF*** -R mutants indicates that lack of SOCS3 ***recruitment*** may contribute to the hyperproliferative phenotype of DELTA715 expressing cells. We therefore looked in more detail at the role of SOCS3 in DELTA715 signaling. SOCS3 is a major transcriptional target of STAT3. Because G-CSFR-DELTA715 lacks two STAT3 activation mechanisms, we performed real time quantitative RT-PCR on bone marrow cells of gcsfr-DELTA715 mice and wild type littermates. We found that SOCS3 transcript levels are significantly reduced in DELTA715 compared to WT animals. As expected, based on the fact that SCN-derived G-CSFR mutants lack Y729, G-CSFR-DELTA715 also showed reduced responsiveness to SOCS3-mediated inhibition. However, while SOCS3 provided considerable residual inhibition of m0 at higher SOCS3 concentrations, G-CSFR-DELTA715 remained completely refractory to the effects of SOCS3, suggesting a tyrosine independent mechanism of action involving the receptor C-terminus. Next to the direct inhibitory effects via SH2 domains, SOCS proteins may downregulate signaling via the SOCS-box which targets internalized receptors for proteosomal degradation. G-CSFR-DELTA715 is severely hampered in ligand-induced endocytosis and may therefore be refractory to this mechanism. Experiments to test this possibility using a mutant of SOCS3 lacking the SOCS-box are ongoing. In conclusion, our data show that C-terminal truncations decrease negative feedback by SOCS3 in three ways: reduced upregulation of SOCS3, lack of the major SOCS3 ***recruitment*** site Y729 and loss of the C-terminus with motifs required for internalization and additional tyrosine independent ***recruitment*** of SOCS3. The lack of appropriate negative feedback by SOCS3 therefore likely contributes to the hyperproliferative phenotype of truncated G-CSFR mutants found in SCN
SO Blood (***November 16 2002***) Vol. 100, No. 11, pp. Abstract No. 2850. print.
Meeting Info.: 44th Annual Meeting of the American Society of Hematology. Philadelphia, PA, USA. December 06-10, 2002. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.

L8 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2008 ACS on STN
TI Oncostatin M regulates the synthesis and turnover of gp130, leukemia inhibitory factor receptor .alpha., and oncostatin M receptor .beta. by distinct mechanisms
AB The cytokine receptor subunits gp130, leukemia inhibitory factor receptor alpha (LIFR alpha) and oncostatin M receptor beta (OSMR.beta.) transduce OSM signals that regulate gene expression and cell proliferation. After ligand binding and activation of the Janus protein tyrosine kinase/STAT and mitogen-activated protein kinase signal transduction pathways, neg. feedback processes are ***recruited***. These processes attenuate receptor action by suppression of cytokine signaling and by down-regulation of receptor protein expression. This study demonstrates that in human lung ***fibroblasts*** or epithelial cells, OSM first decreases the level of gp130, LIFR alpha, and OSMR beta, by ligand-induced receptor degrdn. and then increases the level of the receptors by enhanced synthesis. The transcriptional induction of gp130 gene by OSM involves STAT3. Various cell lines expressing receptor subunits to the different interleukin-6 class cytokines revealed that only LIFR alpha degrdn. is promoted by activated ERK and that degrdn. of gp130, OSMR beta, and a fraction of LIFR alpha involves mechanisms that are gen. from signal transduction. These mechanisms include ligand-mediated dimerization, internalization, and endosomal/lysosomal degrdn. Proteosomal degrdn. appears to involve a fraction of receptor subunit proteins that are ubiquitinated independently of ligand binding.
SO Journal of Biological Chemistry (***2001***), 276(50), 47038-47045
CODEN: JBCHA3; ISSN: 0021-9258

L8 ANSWER 6 OF 11 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI Bacterial stimulation of ***G*** - ***GSF*** expression contributes to PMN persistence at the site of infection
AB Bacterial interactions with airway epithelial cells result in the ***recruitment***, and activation of PMNs through ligation of glycolipid receptors, stimulation of MAP kinases, NF-kappaB and the transcription of proinflammatory cytokines and chemokines. In the studies described, we demonstrate that P. aeruginosa stimulates epithelial ***G*** - ***GSF*** expression which then serves to promote PMN survival. ***G*** - ***GSF*** is produced by macrophages, lymphocytes and ***fibroblasts*** in response to bacterial stimuli although its function in the airway is less well characterized. To determine if ***G*** - ***GSF*** contributes to airway inflammatory responses, the expression of ***G*** - ***GSF*** was monitored in several airway epithelial cell lines following stimulation by P. aeruginosa, S. aureus and selected mutants. There was no constitutive ***G*** - ***GSF*** expression as detected by RT-PCR, but 24 hours following exposure to either organism there was a significant amount of mRNA and a 10-fold increase in ***G*** - ***GSF*** production as measured by ELISA. The in vitro studies were confirmed by demonstration of ***G*** - ***GSF*** by immunocytochemistry of the bronchial epithelial cells in sections of mouse lung 24 hours following infection with either S. aureus or P. aeruginosa. The physiological effects of ***G*** - ***GSF*** on PMN apoptosis were monitored using a PMN viability assay to compare the survival of human PMNs incubated in conditioned media harvested from epithelial cells stimulated with P. aeruginosa or from controls. There was a 75% increase in PMN survival under conditions in which the PMNs were exposed to media from P. aeruginosa stimulated epithelial cells as compared with controls. This effect was negated by the addition of anti- ***G*** - ***GSF*** but not anti-GM-CSF. The results suggest that ***G*** - ***GSF*** transcription is upregulated in airway epithelial cells upon stimulation with P. aeruginosa, and that the secreted protein promotes PMN viability.
SO Abstracts of the General Meeting of the American Society for Microbiology, (***2001***) Vol. 101, pp. 115. print.

Meeting Info. : 101st General Meeting of the American Society for Microbiology Orlando, FL, USA. May 20-24, 2001. American Society for Microbiology
TSSN: 1060-2011.

L8 ANSWER 7 OF 11 MEDLINE on STN DUPLICATE 1
TI Bleomycin stimulates lung ***fibroblasts*** to release neutrophil and monocyte chemotactic activity
AB We determined whether human lung ***fibroblasts*** might release chemotactic activity for neutrophils (NCA) and monocytes (MCA) in response to bleomycin. The human lung ***fibroblasts*** supernatant fluids were evaluated for chemotactic activity by a blind well chamber technique. Human lung ***fibroblasts*** released NCA and MCA in a dose- and time-dependent manner in response to bleomycin. Checkerboard analysis of supernatant fluids revealed that both NCA and MCA were chemotactic. Partial characterization revealed that NCA was partly heat labile, trypsin sensitive, and predominantly ethyl acetate extractable. In contrast, MCA was partly trypsin sensitive and ethyl acetate extractable. The release of chemotactic activity was inhibited by lipoxygenase inhibitors and cycloheximide. Molecular sieve column chromatography revealed that both NCA and MCA had multiple chemotactic peaks. NCA was inhibited by leukotriene B4 receptor antagonist and anti-IL-8 and ***G*** - ***CSF***. Abs. MCA was attenuated by leukotriene B4 receptor antagonist and monocyte chemoattractant protein-1, GM-CSF, and TGF-beta Abs. Leukotriene B4 receptor antagonist and these Abs inhibited the corresponding m.w. chemotactic activity separated by column chromatography. The concentrations of IL-8 ***G*** - ***CSF***, monocyte chemoattractant protein-1, GM-CSF, and TGF-beta in the supernatant fluids significantly increased in response to bleomycin. These data suggest that lung ***fibroblasts*** may modulate inflammatory cell ***recruitment*** into the lung by releasing NCA and MCA in response to bleomycin.
SO Journal of immunology (Baltimore, Md. : 1950), ***(1999 May 15)*** Vol. 162, No. 10, pp. 6200-8
Journal code: 2985117R. ISSN: 0022-1767.

L8 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2008 ACS on STN
TI Expression of multiple angiogenic cytokines in cultured normal human prostate epithelial cells: predominance of vascular endothelial growth factor
AB The cytokines that regulate angiogenesis in normal and malignant prostate tissue are not well studied. Using an RT-PCR-based screen, the authors obsd. that cultured, low-passage normal human prostate epithelial cells (PrECs) express a variety of cytokines which have been shown to have angiogenic and/or endothelial cell-activating properties in various systems. These include vascular endothelial growth factor (VEGF), basic fibroblastic growth factor (bFGF), transforming growth factor-.alpha. (TGF-.alpha.), transforming growth factor-.beta. (TGF-.beta.), interleukin-8 (IL-8), tumor necrosis factor-.alpha. (TNF-.alpha.), granulocyte-macrophage colony-stimulating factor (GM-CSF), and granulocyte colony-stimulating factor (***G*** - ***CSF***). Expression of VEGF, bFGF, GM-CSF, ***G*** - ***CSF***, TGF-.alpha., and TNF-.alpha. in these cells was confirmed by immunohistochem. Culture medium conditioned by normal human PrECs for periods of up to 96 h were found to contain VEGF, GM-CSF, ***G*** - ***CSF***, IL-8, TGF-.beta.1, and TGF-.beta.2 but not TNF-.alpha. or bFGF, as detd. by ELISA. Of these, VEGF was by far the most prominently expressed angiogenic cytokine (approx. 2500 pg/mL conditioned medium at 96 h vs. 30 to 100 pg/mL conditioned medium for the other cytokines). PrEC-conditioned medium induced an approx. 2-fold stimulation of [3H]-thymidine incorporation in cultured human umbilical cord endothelial cells (HUVECs) deprived of the endothelial growth factors VEGF and bFGF; this stimulation was abolished

by neutralizing antibodies directed against VEGF but not bFGF, IL-8, GM-CSF or TNF-alpha. VEGF expression by PrECs was not markedly altered by administration or deprivation of other angiogenic cytokines for which these cells have receptors, suggesting that there is not a hierarchy of cytokines controlling its expression; however, retinoic acid, a component of PrEC growth medium, was found to modestly suppress VEGF at physiol concns. (1 ng/mL). These data suggest that normal PrECs express a variety of angiogenic cytokines, most prominently VEGF, to ***recruit*** a supporting vasculature, even in culture. The authors' data also suggest that the ability of malignant PrECs to stimulate angiogenesis may be intrinsic and does not need to be acquired during oncogenesis.

CS International Journal of Cancer (***1999***), 80(6), 868-874
CODEN: IJCNAW; ISSN: 0020-7136

L8 ANSWER 9 OF 11 MEDLINE on STN DUPLICATE 2

TI Enhanced detection, maintenance, and differentiation of primitive human hematopoietic cells in cultures containing murine ***fibroblasts*** engineered to produce human steel factor, interleukin-3, and granulocyte colony-stimulating factor.

AB To determine whether the sensitivity of the human long-term culture-initiating cell (LTC-IC) assay could be increased, we have evaluated a spectrum of different ***fibroblast*** cell lines for their abilities to influence the number of cells detectable as LTC-IC, to influence LTC-IC maintenance, and/or to influence LTC-IC differentiation into colony-forming cells (CFC) in cocultures containing various sources of LTC-IC. In a series of initial experiments with highly purified subpopulations of CD34+ cells from normal human marrow, no significant difference could be found between any of 3 different murine stromal ***fibroblast*** cells in terms of their support of either LTC-IC detection (CFC production) or maintenance (over a 6-week period), and all were equivalent to primary human marrow feeders (HMF). On the other hand, murine M2-10B4 ***fibroblasts*** engineered to produce high levels of both human granulocyte colony-stimulating factor (***G*** - ***GSF***) and interleukin-3 (IL-3; 100 and 4 ng/mL, respectively), either alone or mixed 1:1 with ST/SI ***fibroblasts*** engineered to produce high levels of soluble Steel factor (SF), with or without production of the transmembrane form of SF (60 and 4 ng/mL, respectively), stimulated the production of up to 20-fold more CFC in LTC of cells from normal human marrow, ***G*** - ***GSF*** -mobilized blood or cord blood when compared with parallel cocultures containing HMF. Limiting dilution analysis of the CFC output from all three sources of LTC-IC showed that most of this increase was due to an ability of the engineered feeders to increase the plating efficiency of the LTC-IC assay (approximately 14-fold for marrow LTC-IC and approximately 4-fold for cord blood or mobilized blood LTC-IC). Analysis of the phenotype of these additionally ***recruited*** LTC-IC from marrow showed they had the same primitive CD34+CD45RA-CD71- phenotype as conventionally defined LTC-IC. The limiting dilution studies also showed that the average number of CFC produced per LTC-IC was additionally and independently increased to yield values of 18 CFC per LTC-IC in marrow, 28 for LTC-IC in cord blood, and 25 for LTC-IC in ***G*** - ***GSF*** -mobilized blood. Replating of cells from primary LTC with different feeders into secondary LTC-IC assays containing the best combination of engineered feeders showed that LTC-IC maintenance could be significantly enhanced (up to 7-fold as compared with primary cocultures containing HMF). However, this enhancement was still not sufficient to amplify the number of LTC-IC present after 6 weeks above the input value. Thus, engineering murine ***fibroblasts*** to produce sufficient SF ***G*** - ***GSF*** , and IL-3 can markedly enhance the detection as well as the maintenance in vitro of a very primitive population of human progenitor cells present in normal adult marrow, mobilized blood, and cord blood by providing the most sensitive assay conditions thus far described. The present findings also provide

new evidence of biologic heterogeneity between different cell populations that can be operationally identified as LTC-IC, thus re-emphasizing the importance of limiting dilution analyses to distinguish between quantitative and qualitative effects on these cells.

SO Blood ***(1996 Nov 15)*** Vol. 88 No. 10, pp. 3765-73.
Journal code: 7603509. ISSN: 0006-4971.

L8 ANSWER 10 OF 11 MEDLINE on STN

TI Microenvironmental influences on inflammatory cell differentiation.

AB Airways inflammation involves accumulation of inflammatory cells such as eosinophils, basophils and mast cells, which are derived from progenitors in marrow and blood. The inflamed tissue of the airways, through its structural (epithelium, stromal) and inflammatory cell components, produces an array of cytokines which can influence the differentiation of inflammatory cell progenitors. It is particular mechanism that we have investigated, showing that molecules such as GM-CSF ***G*** - ***CSF*** IL-6, IL-8 and SCF can be produced by airways epithelial cells and ***fibroblasts*** in quantities sufficient to induce hemopoietic events, either systemically or locally. Corticosteriods may act therapeutically, at least in part, to block inflammatory cell differentiation, and thus ***recruitment*** , into the allergic inflammatory process in the airways.

SO Allergy ***(1995)*** Vol. 50 No. 25 Suppl, pp. 25-8. Ref: 20
Journal code: 7804028. ISSN: 0105-4538.

L8 ANSWER 11 OF 11 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

TI CYTOKINES AND FUNGAL INFECTIONS

AB Cytokines play a major role within the immune system as necessary mediators involved in the set up of the specific immune response as well as factors which amplify the anti-microbial response. It includes activation of B and T lymphocytes leading to the production of antibodies and generation of specific cytotoxic T cells, activation of phagocytic cells, ***recruitment*** of cells at the infectious site, and stimulation of the hematopoiesis leading to an increased number of active cells. Furthermore, cytokines are a link between immune cells and many others of the organism. For example, some of them act on the central nervous system and possess pro-inflammatory activities. During fungal infections, monocytes and macrophages are triggered either by fungi or their derived products and release tumor necrosis factor alpha [TNF alpha] and interleukin-1 [IL-1]. Other cells such as ***fibroblasts*** NK cells, and neutrophils can be activated by fungi to produce cytokines. In addition, T lymphocytes are also stimulated and release interferon gamma [IFN gamma], a potent macrophage activator. If an excess of cytokines is produced, it is then possible to detect them in plasma. This has been reported for TNF alpha, IL-3 and IL-6. The capacity of these cytokines to enhance in vitro anti-fungal activities of phagocytic cells has been reported for IFN gamma, IL-1 alpha, and macrophage-colony stimulating factor [M-CSF] which amplify the monocytes/macrophages killing activities, and for IFN gamma, and IL-8 which trigger the functions of neutrophils. In the mouse model, in vivo experiments have demonstrated the efficiency of IL-1, M-CSF ***G*** - ***CSF*** and TNF alpha, to increase the protection against fungal infections. In addition, some cytokines, like IL-1, can act synergistically with anti-fungi drugs. The efficiency of these cytokines are often associated with a pre-treatment before injection of the microbial agent. Thus, these animal models are not always reliable for the treatment of human fungal infections. Th1 as well as Th2 [two T lymphocytes subpopulations which differ in their pattern of cytokine production] can be activated during experimental fungal infection and led to different immunity associated or not with protection. Further knowledge of the immune response to fungal infections as well as a better

understanding of the relative role of individual cytokines to induce protection will help in the near future to enhance the defense mechanisms involved during fungal infections.

SO Journal de Mycologie Medicale, (***1992***) Vol. 2, No. 2, pp. 61-67. ISSN: 1156-5233.

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